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Cardiac Valve Replacement

The Rationale for Earlier Operation

JOSEPH S. CAREY, MD, WILLIAM G. PLESTED, MD, *and* CALVIN SCOTT, BS, *Los Angeles*

BECAUSE OF THE HIGH RISK of cardiac valve replacement, operation is usually delayed until incapacitating (functional class III) symptoms develop. Since 1966, however, operative mortality has decreased to approximately 5 percent in many reported series (Table 1). In addition, improved prosthetic materials have significantly reduced valve-related complications. The most common cause of death after cardiac valve replacement has been myocardial failure rather than dysfunction of the prosthetic device.[1-6] Under ideal conditions, such as the absence of severe myocardial failure, the risk of valve replacement should be relatively low.

At Wadsworth Veterans Administration Hospital, single or double valve replacement was performed in 138 consecutive patients from October 1, 1966 through October 1, 1972. Although all but a few patients had functional class III or IV disease, and many were operated upon in emergency, the hospital mortality was low (Table 1). Four of the five hospital deaths were due to preventable technical errors. These results suggest that the risk of cardiac valve replacement may indeed be lower than it is commonly thought to be. An analysis of the late results in this series of patients, all of whom have now been followed at least one year, indicates that functional rehabilitation is frequently not achieved. The relatively poor long-term results suggest that delaying operation may inhibit functional rehabilitation.

This report examines the operative techniques which were used to obtain the gratifying early results. In addition, the long-term functional results are analyzed, and we question the rationale of delaying operation until patients reach functional class III status.

From the Thoracic Section, Surgical Service, Veterans Administration Wadsworth Hospital Center, and the UCLA School of Medicine, Los Angeles.

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Reprint requests to: J. S. Carey, MD, Santa Monica Medical Plaza, 1260 Fifteenth St., Santa Monica, CA 90404.

Cardiac valve replacement (single or double) was carried out in 138 consecutive patients with valvular heart disease over a six-year period at the Wadsworth Veterans Administration Hospital. All but a few had functional class III or IV disease. Hospital mortality was 3.6 percent.

Normothermia, coronary perfusion and beating of the heart were maintained throughout the procedure. All patients were followed at least one year and the average follow-up was four and a half years. The late mortality was 27 percent. Seventy-five percent of the late deaths were due to progressive cardiac disease. The mortality due to prosthetic valve dysfunction was 4 percent.

Subjective evaluation of survivors revealed that a high percentage remained symptomatic. Only 50 percent of the total group of patients were significantly improved.

The high incidence of late mortality and poor long-term functional results are contrasted to the low operative risk and improved reliability of prosthetic valves. The rationale of delaying operation until functional class III limitation develops is questioned. Earlier operation is recommended before irreversible myocardial hypertrophy and fibrosis develop.

Clinical Material

All patients undergoing cardiac valve replacement for valvular heart disease operated upon by the authors between October 1, 1966, and October 1, 1972, were included in the series. In no case was operation withheld because of severity of disease. Patients undergoing cardiac valve replacement together with correction of associated lesions (aortic aneurysm, coronary artery stenosis, congenital lesions) were not included. All patients had functional class III or class IV disease (New York Heart Association) at the time of operation, except for three who underwent replacement of a previously implanted prosthetic device for multiple embolic episodes. All but two were men. Clinical data revealed that 79 percent were or had been cigarette smokers and the majority were social users of alcohol. The average age at the time of operation was 48 years. In most instances, the patients had been followed for many years in the Cardiology Clinic. Operation was done when incapacitating symptoms which were refractory to medical therapy developed. The decision to operate was made at a conference of the same group of cardiologists and surgeons throughout the study period.

Surgical Technique

Cardiopulmonary bypass was carried out with an Olson pump and a disposable bubble oxygenator (Bentley Laboratories, Santa Ana, California), except in a few cases early in the series in which a rotating disc (Kay-Cross) oxygenator was used. Left ventricular vents and normothermic perfusion were used in all cases. The oxygenator was primed with a mixture of balanced electrolyte solution and colloid (usually whole blood and albumin).

Aortic valve replacement was performed through a median sternotomy using continuous normothermic coronary perfusion, permitting the heart to beat throughout the procedure. The aorta

TABLE 1.—*Data on Hospital Mortality in Various Series of Cardiac Valve Replacement*

Reference	*Time Period*	*No. of Patients*	*Valve Replaced*	*Prosthetic Valve*	*Hospital Mortality (percent)*
Messmer[1]	1969-1971	460	Aortic	Bjork-Shiley	8.5
Henze[2]	1969-1972	161	Aortic	Bjork-Shiley	5.0
Behrendt[3]	1971	252	Aortic, Mitral, Double	Starr-Edwards	5.5
Griepp[4]	1971-1972	146	Aortic, Mitral, Double	Starr-Edwards	4.7
Pacifico[5]	1967-1970	304	Aortic	Starr, Homograft	6.2
Cooley[6]	1971-1972	138	Mitral, Mitral and Aortic	Cooley-Cutter	5.8
Present Series	1966-1972	138	Aortic, Mitral, Double	Starr-Edwards	3.6

was opened through a transverse or slightly oblique aortotomy without entering the coronary sinus. Coronary perfusion catheters (Mayo balloons) were anchored in place with one 5-0 suture through the aortic wall. In calcified aortic valves no effort was made to remove calcium deposits located in the aortic or myocardial wall. In such instances heavy sutures were placed through the calcium and then into the prosthesis. Interrupted figure-8 horizontal mattress sutures of 2-0 Tevdek, 4 to 6 in each valve cusp, were used to anchor the prosthetic valve in place. Starr-Edwards valves were used for aortic valve replacement in all instances. From November 1967 on, only cloth-covered models have been used. The aorta was closed with a continuous 4-0 Tevdek suture leaving the coronary perfusion catheters in place as long as possible. A Foley catheter was passed through the valve into the left ventricle during aortic closure. At the uppermost point of aortic closure, a final mattress suture was placed around the coronary catheters. At this point the left ventricular vent was clamped and the heart allowed to fill with blood. The coronary perfusion catheters were then removed and the remainder of the aortic incision closed. As soon as the aortotomy was closed the aortic cross clamp was removed. Using this technique, coronary perfusion was interrupted only for the minute or two necessary to close the remainder of the aortotomy incision. The patient was then immediately weaned from cardiopulmonary bypass.

When both aortic and mitral valve replacement were performed, coronary perfusion was instituted at the beginning of the procedure and continued during the replacement of both valves.

Mitral valve replacement was also performed through a median sternotomy. After institution of cardiopulmonary bypass, the aorta was cross-clamped and the left atrium was opened through an incision in the posterior inter-atrial groove. The anterior leaflet of the mitral valve was incised and the anterior row of sutures was placed while traction was held on the partially excised valve. The aortic cross-clamp was removed after 10 minutes, and the sutures were placed into the prosthetic valve. The natural valve was then completely excised and the posterior row of sutures was placed, with another brief period of aortic cross-clamping if necessary. Figure of 8 mattress sutures of 2-0 Tevdek were used to fix the valve in place. Starr-Edwards prosthetic valves were used in all instances for mitral valve replacement. After January 1968, only cloth-covered models were used. During closure of the atriotomy incision, a Foley catheter was left in place to keep the prosthetic valve inoperative. When closure of the atriotomy was completed, the catheter was removed and all air evacuated from the heart through the apex of the left ventricle by way of the left ventricular vent.

The tricuspid valve was also approached through a median sternotomy. Valve replacement was accomplished by techniques similar to those employed for mitral valve replacement. However, only Bjork-Shiley prosthetic valves have been implanted in the tricuspid position.

Postoperative Care

All patients were monitored in the intensive care unit for 3 to 5 days after operation. Standard variables were monitored during the first few postoperative days, including arterial pressure, central venous pressure, arterial blood gases, blood volume and hourly urine output. Left atrial pressure measurements were not employed. Because of the use of hemodilution perfusion, fluid intake was kept at a minimum during the first few postoperative days and urine output was maintained with furosemide or ethacrynic acid. The liberal use of these diuretic agents has greatly reduced need for tracheotomy in the postoperative period. It is, however, important to replace potassium losses caused by the use of diuretic drugs. We have

TABLE 2.—*Death in Hospital and Late Mortality after Starr-Edwards Valve Replacement*

	No. Pts.	Hosp. Mort.	Late Mort.
Single valve replacement			
Aortic	66	1	16
Mitral	55	3	15
Tricuspid	1	0	0
Total	122	4(3.3%)	31(25%)
Multiple valve replacement			
Aortic and mitral	13	1	4
Mitral and tricuspid	3	0	1
Total	16	1(6.3%)	5(31%)
Combined single and multiple	138	5(3.6%)	36(27%)

TABLE 3.—*Causes of Hospital Mortality*

Procedure Valve Replacement	Cause of Death
1. Aortic	Mesenteric infarction
2. Aortic and mitral	Right coronary artery dissection
3. Mitral	"Low cardiac output"—Prosthesis too large
4. Mitral	Retrograde aortic dissection
5. Mitral	Postoperative cardiac tamponade

found that the urinary concentration of potassium averages 40 to 50 mEq per liter during diuresis induced by ethacrynic acid.

Methods of Data Review

Medical information was digitized on data transcription sheets, and computer analysis was performed at the Western Research Support Center of the Veterans Administration (Sepulveda, California). Follow-up data were obtained by questionnaires sent to patients or by direct contact with the family or physicians. Causes of death were determined from clinical data or autopsy information.

Questionnaires were returned by 94 of 97 survivors. Information was obtained from relatives or clinical records on the 3 remaining patients. One patient was lost to follow-up nine months after operation. Functional class was determined by evaluating the patient's ability to climb stairs and walk level distances, and by symptoms and subjective sense of improvement. If the patient's activity was essentially unlimited and he was asymptomatic or mildly symptomatic and (subjectively) greatly improved, he was considered to be Class I. A patient who could not climb more than two flights of stairs or walk two level blocks without stopping but was minimally symptomatic and felt greatly improved was considered Class II. A patient who was limited to the same functional activity but was moderately symptomatic and considered himself only slightly improved was reported as Class III.

Results

Survival

The hospital mortality was 3.3 percent for single valve replacement, and the late mortality was 25 percent; for multiple valve replacement, 6.3 and 31 percent respectively (Table 2).

The causes of hospital death are listed on Table 3. All deaths occurred postoperatively; there was no patient who could not be removed from cardiopulmonary bypass. Among 79 patients undergoing aortic valve replacement alone or in combination with mitral or tricuspid replacement, there were 2 hospital deaths: one was caused by technical errors in the use of coronary perfusion and the other was caused by diffuse mesenteric infarction which occurred after isolated aortic valve replacement for aortic insufficiency. Three patients died after mitral valve replacement; all three deaths were due to technical errors occurring at the time of operation. In one patient who died of "low cardiac output," indentations from the cage of the prosthetic valve and hematoma formation were found on the interventricular septum at autopsy, indicating the prosthetic device was too big. Retrograde aortic dissection occurred after institution of emergency cardiopulmonary bypass in another patient; although bypass was reinstituted through the ascending aorta, cerebral damage occurred which eventually led to the patient's death. The third death was caused by cardiac tamponade occurring 10 days postoperatively which was erroneously treated as pulmonary embolism.

The causes of late death are listed by categories in Table 4. There were no definite valve-related deaths after aortic valve replacement, but four late valve-related deaths occurred after mitral valve replacement, and one after aortic and mitral replacement. There were two instances of thrombus formation found at autopsy on a prosthetic mitral valve, which probably led to the patients' death; one occurred in a patient with a Model 6500 series (low-profile) Starr-Edwards valve, and the other in a patient with a 6300 series (early cloth-covered model) valve. Neither of these devices are currently in use. A third patient died of a splenic hemorrhage probably caused by embolism. A model 6120 (silastic ball) prosthetic valve was

TABLE 4.—*Causes of Late Death 3 Months to 6.3 Years (Average 2.3 Years) after Hospital Discharge*

	Aortic	Mitral	Multiple	Total	Autopsy
Prosthetic failure	0	4	1	5	5
Non-cardiac	3	4	1	8	8
Progressive cardiac disease	7	2	2	11	8
Unknown	6	5	1	12	1
Total	16	15	5	36	21(60%)

TABLE 5.—*Significant Non-Fatal Postoperative Complications During Hospitalization*

	Aortic	Mitral	Multiple	Total
Arrhythmia	27	29	7	63(46%)
Respiratory	8	11	2	21(15%)
Tracheostomy required	4	6	4	14(10%)
Low cardiac output "syndrome"	5	3	5	13(9%)
Stroke with residual	4	0	1	5(4%)

TABLE 6.—*Significant Valve-Related Complications*

	Aortic	Mitral	Multiple	Total
Mortality	0	5	1	6(4.1%)
Reoperation	2	2	1	5(3.6%)
Stroke with residual	6	3	0	9(6.5%)

TABLE 7.—*Subjective and Functional Results in Survivors at the End of the Current Follow-up Period*

	No. of Patients	Percent of Survivors
Subjective result		
Greatly improved	39	40
Slightly improved	28	29
Not improved or worse	20	21
Unknown	10	10
Functional result		
Class I (NYHA)	31	32
Class II	38	39
Class III	27	28
Class IV	0	0
Lost to follow-up	1	1

used in this patient. The fourth valve-related death also occurred in a patient with a 6120 series valve, in whom periprosthetic leak occurred. The fifth value-related death occurred after aortic and mitral replacement with model 2300 (aortic) and 6300 (mitral) prosthetic valves. Several patients died of non-cardiac causes, including bowel obstruction, malignant disease, and trauma. The most common cause of late death was "progressive cardiac disease." In most of these patients, clinical and autopsy findings indicated normal functioning of the prosthetic valve. A significant number of patients also died of undetermined causes. In these cases, clinical and autopsy information was not adequate to clearly determine the cause of death.

Postoperative Complications

Postoperative complications occurred in the majority of patients (Table 5). Some type of arrhythmia occurred in nearly half of the survivors. Significant respiratory complications occurred in 15 percent, and tracheostomy was required in 10 percent. Low cardiac output "syndrome" (poor peripheral perfusion, low urine output, vasopressors required) occurred in 9 percent of patients. Five patients had neurological damage upon awakening from anesthesia after aortic or aortic and mitral valve replacement. The neurological damage was major (accompanied by paralysis) in only one of the five. Although these strokes are thought to be due to calcium or air embolism, the possibility of embolism from the prosthetic valve cannot be ruled out.

Prosthetic Valve-Related Complications

All the known valve-related complications are listed in Table 6. There were one early and four late valve-related deaths after mitral valve replacement, and one late valve-related death after aortic and mitral replacement (discussed above). All six occurred with prosthetic valves now no longer in use (models 6120, 6500, 6300, and 2300). Five patients required reoperation, three for periprosthetic leak (two aortic, one mitral) and two for removal of early model cloth-covered prostheses which were thought to be obstructive (one aortic, one mitral). There were nine clear episodes of postoperative embolism with neurological residual: eight of the patients have minor weakness or memory loss, and one is bedridden with hemiplegia. All episodes occurred in patients with earlier model prosthetic valves.

Functional Results in Survivors

As determined by subjective and functional results 69 percent of patients were improved and 71 percent were in Class I or Class II (Table 7). A relatively large proportion of patients in the current series felt "not improved or worse," and 28 percent were in functional Class III postoperatively.

Discussion

The occurrence of myocardial failure of uncertain origin in the early postoperative period after cardiac valve replacement has been noted by

several investigators. Various types of "myocardial necrosis" have been described, including a concentric type of subendocardial necrosis,[7] and a diffuse, patchy distribution of hemorrhage and necrosis.[8] The absence of these findings in the present series suggests that some aspect of surgical techniques we used may have prevented the development of myocardial necrosis. The most likely explanation is that our use of normothermic coronary perfusion, allowing the heart to beat throughout the operative procedure, either minimized or eliminated the occurrence of myocardial damage during operation. There is evidence that the use of electrical fibrillation for prolonged periods may produce myocardial hemorrhage and necrosis.[9] In addition, spontaneous fibrillation, which may occur during hypothermic coronary perfusion, may also damage the myocardium. Ischemic arrest may be well tolerated for 30 to 45 minutes in the normal heart, but the hypertrophied heart tolerates ischemia poorly, as was indicated by experimental evidence in dogs.[10] Cooley and coworkers[11] reported an operative mortality of 52 percent in patients in whom ischemic arrest was employed during aortic and mitral valve replacement for aortic stenosis and mitral stenosis. Kirklin's group,[5] on the other hand, reported an operative mortality of 6.2 percent after aortic valve replacement procedures in which mild hypothermia, coronary perfusion and a beating heart were used. Topical hypothermia has also been associated with low hospital mortality, particularly using cold saline rather than ice in the pericardial well.[5,12]

The patients in this series were drawn from a relatively fixed hospital population so that preselection, such as may occur in a referral practice, is not a factor in determining the quality of clinical material. In many instances, Veterans Administration Hospital patients are poorer surgical risks because of delay in obtaining medical care, resulting in the development of advanced disease before they come to the hospital. In addition, most male veterans are heavy smokers and users of alcohol. No patient was refused operation, however, because of severity of disease or because of the presence of high surgical risk.

In spite of the absence of pre-selection and the inclusion of all patients as surgical candidates regardless of "risk," the hospital mortality for this series is lower than that reported from other major centers. This is strong evidence that the use of normothermic coronary perfusion, and the avoidance of prolonged ischemic arrest and electrical or spontaneous fibrillation, provides the best "myocardial protection" during cardiac valve replacement.

The high mortality and sub-optimal subjective and functional results obtained in this series are disconcerting. However, very little late morbidity seems to be directly related to the prosthetic valves. Six of the 16 valve-related complications were due to technical errors (selection of too large a prosthetic valve, periprosthetic leak), and the majority of the remaining complications occurred in patients with earlier model valves. Current data indicate that the present model Starr-Edwards valves (2310-2320 aortic, 6310-6320 mitral) are hemodynamically satisfactory.[13] These models were used in the last 48 patients of this series, and no definite valve-related complications have occurred in this group.*

Most of the late mortality in this series was related to progressive cardiac disease. This suggests that operation may have been performed too late in the course of the disease. Irreversible congestive heart failure and ventricular hypertrophy may already be present at the time of operation. In spite of moderate improvement by valve replacement, symptoms eventually recur; in such patients, the clinical condition may temporarily stabilize, but deterioration then occurs. Other patients may die suddenly of unknown causes within the first two years. In most such cases there was pronounced left ventricular hypertrophy or dilatation at the time of operation. Although it is possible that death in some of these cases may have been related to prosthetic failure, progression of irreversible myocardial failure seems to be a more likely possibility.

In order to improve late mortality as well as functional results, earlier operation would seem to be indicated in some patients, particularly those with aortic valve disease or mitral regurgitation in whom severe, perhaps irreversible, left ventricular failure is likely to develop at any time. In a previous study, we found that the late mortality of mitral stenosis was considerably less than that of mitral regurgitation.[14] This finding, which has been reported by other investigators,[15] also suggests that

*As of April 1, 1974, 100 patients had had replacement of aortic or mitral valves, or both, using current model Starr-Edwards devices. One hospital death was definitely valve-related (cerebral-embolus), and autopsy evidence of mild thrombus formation was found in a second patient who died of chronic congestive heart failure. Three late deaths occurred suddenly and autopsy was not performed. No evidence of embolism, thrombus formation, infection, periprosthetic leak or cloth wear has been found in the remaining patients in this series. Definite valve-related complications have thus occurred in 2 percent of patients receiving current model cloth-covered Starr-Edwards valves.

earlier operation should be performed in patients with left ventricular overload.

Future patients may be benefited by more precise studies of ventricular function. For example, the routine use of non-invasive studies such as echocardiography may allow the identification of critical increases in left ventricular mass before the development of irreversible cardiac failure.

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